Current Status of Provocative Food Testing

PROVOCATIVE FOOD TESTING was revived and popularized by Rinkel and Lee in 1960, and the technique has had numerous proponents since that time. The method, however, remains controversial and is not generally used in the diagnosis of food (or other) allergy by most allergists. The testing procedure consists of the intradermal injection of a food extract of potency and volume sufficient to induce systemic symptoms, followed by the immediate injection of a small amount of the same food extract to neutralize the symptoms. Duke, in 1921, described a similar procedure in which food extracts were used to produce symptoms in patients with gastrointestinal and genitourinary complaints followed by neutralization of the symptoms with epinephrine. A recent modification of this procedure utilizes sublingual administration of food extracts or other materials. Advocates of these methods claim an accuracy of up to 85 percent in identifying specific food sensitivity. Such advocates also state it is imperative that the patient have complete evaluation of inhalant allergy to pollens, dust, molds, and epidermals, and that environmental allergen control is essential before food testing.

Long overdue objective scientific evaluation of these provocative-neutralizing techniques is now being performed. The validity of the technique was not established in one recent study of 20 patients with food allergy.

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Inhibition of Histamine Release by Cyclic 3', 5'-Adenosine Monophosphate (CAMP)

THERE IS MUCH REASON to believe that exposure to antigen produces allergic symptoms, such as asthma and allergic rhinitis, by stimulating the release of histamine and slow reacting substance of anaphylaxis (SRS-A) from IgE-sensitized blood basophiles or tissue mast cells at the target or shock organ sites.

In examining the mechanism involved in the antigenic release of histamine and SRS-A, it has been learned that materials such as isoproterenol, theophylline, dibutyryl CAMP, and prostaglandins E_1 and E_2 which mimic or increase the cellular content of CAMP also inhibit the release of histamine and SRS-A from human lung tissue or leukocytes. Other leukocytic functions, such as the killing of target cells by lymphocytes and lymphocytic transformation, are also inhibited by these materials.

In the many cellular systems in which CAMP has been studied, it usually functions as a "second messenger" to promote a hormone-induced *stimulating* action. The *inhibition* of allergic histamine release by CAMP stands as an interesting contrast.

Further studies of the histamine release mechanism, however, are clearly needed since other materials such as cholinergic agents disodium cromoglycate and diethylcarbamazine, which also inhibit allergic histamine release, apparently have no influence on cellular CAMP levels.

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Status of Testing for Penicillin Allergy

ANAPHYLAXIS and other allergic reactions continue to be a major concern to all those administering and receiving penicillin therapy. Skin tests with penicillin G and several of its derivatives appear to be the simplest and most practical way to predict allergic reactions to penicillin. The major antigenic determinant derived from penicillin G is penicillenic acid which, through the penicilloyl (BPO) radical complexed to body protein, acts as a sensitizing antigen for the stimulation of antibody production. IgE anti-BPO antibodies are thought to be responsible for penicillin-induced urticaria and reactions of serum sickness type. Penicilloyl polylysine (BPO-PPL) skin tests detect these IgE antibodies, while BPO-PPL hemagglutination tests detect IgM and IgG antibodies. Minor antigenic determinants derived from penicillin G, including penicillenate, penamaldate, penicillamine and penicillin G itself can induce IgE antibodies important as causes of anaphylactic reactions. These IgE antibodies may be detected by skin tests with a minor determinant mixture (MDM). Clinical studies have demonstrated that